What is claimed:

1. A method of determining whether a compound inhibits HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a p51 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the p66 subunit polypeptide and the p51 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein a decreased level of activity of the reporter gene in step (a) indicates that the compound inhibits formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and the p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

2. A method of determining whether a compound inhibits formation of a complex between a p66 subunit polypeptide of HIV-1 reverse transcriptase and a p51 subunit polypeptide of HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which

expresses a fusion protein comprising a p51 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the p66 subunit polypeptide and the p51 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein a decreased level of activity of the reporter gene in step (a) indicates that the compound inhibits formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and the p66 subunit polypeptide of HIV-1 reverse transcriptase.

3. A method of determining whether a compound inhibits HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a p51 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the p66 subunit polypeptide and the p51 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein an increased level of activity of

the reporter gene determined in step (a) indicates that the compound is an activator of the formation of the complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and the p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

4. A method of determining whether a compound enhances formation of a complex between a p66 subunit polypeptide of HIV-1 reverse transcriptase and a p51 subunit polypeptide of HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a p51 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the p66 subunit polypeptide and the p51 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein an increased level of activity of the reporter gene determined in step (a) indicates that the compound is an activator of the formation of the complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and the p66 subunit polypeptide of HIV-1 reverse transcriptase.

5. A method of determining whether a compound inhibits HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a first p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a second p66 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the first p66 subunit polypeptide and the second p66 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein a decreased level of activity of the reporter gene in step (a) indicates that the compound inhibits formation of a complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

6. A method of determining whether a compound inhibits formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a first p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid

which expresses a fusion protein comprising a second p66 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the first p66 subunit polypeptide and the second p66 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein a decreased level of activity of the reporter gene in step (a) indicates that the compound inhibits formation of a complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase.

7. A method of determining whether a compound inhibits HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a first p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a second p66 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the first p66 subunit polypeptide and the second p66 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the

reporter gene determined in the absence of the compound, wherein an increased level of activity of the reporter gene in step (a) indicates that the compound is an activator of the formation of the complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

8. A method of determining whether a compound enhances formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase which comprises:

a) contacting a yeast cell with the compound, which cell comprises (i) a first plasmid which expresses a fusion protein comprising a first p66 subunit polypeptide of HIV-1 reverse transcriptase, (ii) a second plasmid which expresses a fusion protein comprising a second p66 subunit polypeptide of HIV-1 reverse transcriptase, and (iii) a reporter gene which is activated in the presence of a complex between the first p66 subunit polypeptide and the second p66 subunit polypeptide, and determining the level of activity of the reporter gene in the cell in the presence of the compound; and

b) comparing the level of activity of the reporter gene determined in step (a) with a level of activity of the reporter gene determined in the absence of the compound, wherein an increased level of activity of the reporter gene in step (a) indicates that the compound is an activator of the formation of the complex between the first p66 subunit polypeptide of

HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase, thereby indicating that the compound inhibits HIV-1 reverse transcriptase.

9. The method of any one of claims 1-8, wherein (a) the fusion protein expressed by the first plasmid comprises a peptide having a DNA binding domain, and (b) the fusion protein expressed by the second plasmid comprises a peptide having a transcription activation domain.

10. The method of claim 9, wherein the DNA binding domain is a LexA DNA binding domain.

11. The method of claim 10, wherein the peptide having a DNA binding domain comprises LexA amino acid residues 1-87.

12. The method of claim 10, wherein the peptide having a DNA binding domain comprises LexA amino acid residues 1-202.

13. The method of claim 9, wherein the DNA binding domain is a GAL4 DNA binding domain.

14. The method of claim 9, wherein the transcription activation domain is a GAL4 transcription activation domain.

15. The method of claim 14, wherein the peptide having the transcription activation domain comprises GAL4 amino acid residues 768-881.

16. The method of claim 9, wherein the transcription activation domain is a VP16 transcription activation domain.

17. The method of any one of claims 1-8, wherein (a) the fusion protein expressed by the first plasmid comprises a peptide having a transcription activation domain, and (b) the fusion protein expressed by the second plasmid comprises a peptide having a DNA binding domain.

18. The method of claim 17, wherein the DNA binding domain is a LexA DNA binding domain.

19. The method of claim 18, wherein the peptide having a DNA binding domain comprises LexA amino acid residues 1-87.

20. The method of claim 18, wherein the peptide having a DNA binding domain comprises LexA amino acid residues 1-202.

21. The method of claim 17, wherein the DNA binding domain is a GAL4 DNA binding domain.

22. The method of claim 17, wherein the transcription activation domain is a GAL4 transcription activation domain.

23. The method of claim 22, wherein the transcription activation domain comprises GAL4 amino acid residues 768-881.

24. The method of claim 17, wherein the transcription

activation domain is a VP16 transcription activation domain.

25. The method of any one of claims 1-8, wherein the fusion protein expressed by the first plasmid, the second plasmid or both plasmids comprises a peptide comprising consecutive alanine residues.

26. The method of claim 25, wherein the peptide comprising consecutive alanine residues comprises at least 6 alanine residues.

27. The method of any one of claims 1-8, wherein the fusion protein comprises an influenza hemagglutinin (HA) epitope tag.

28. The method of any one of claims 1-8, wherein the reporter gene is a LacZ reporter gene.

29. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p66 subunit polypeptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, and an influenza hemagglutinin (HA) epitope tag, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, which influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of

the p51 subunit polypeptide.

30. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p66 subunit polypeptide is bound at it's C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

31. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a LexA peptide corresponding to amino acid residues 1-87, wherein the LexA peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the of the p66 subunit polypeptide; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, and an influenza hemagglutinin (HA) epitope tag, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, which influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

32. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a LexA peptide corresponding to amino acid

residues 1-87, wherein the LexA peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the of the p66 subunit polypeptide; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

33. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a LexA peptide corresponding to amino acid residues 1-202, and a peptide comprising six consecutive alanine residues, wherein the LexA peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

34. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a LexA peptide corresponding to amino acid residues 1-202, and a peptide comprising six consecutive alanine residues, wherein the LexA peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide

comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, and an influenza hemagglutinin (HA) epitope tag, which Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, which influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

35. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, an influenza hemagglutinin (HA) epitope tag, and a peptide comprising six consecutive alanine residues, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, wherein the influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p51 subunit polypeptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain.

36. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, an influenza hemagglutinin (HA) epitope tag, and a peptide comprising six consecutive alanine residues, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, wherein the influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein peptide comprising a LexA protein DNA binding domain is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

37. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, an influenza hemagglutinin (HA) epitope tag, and a peptide comprising six consecutive alanine residues, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, wherein the influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide

comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a Gal4 protein DNA binding domain, which peptide comprising a Gal4 protein DNA binding domain is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

38. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p51 subunit polypeptide is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain.

39. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a LexA protein DNA binding domain, which peptide comprising a LexA protein DNA binding domain is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

40. The method of any one of claims 1-4, wherein (a) the fusion protein expressed by the first plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide; and (b) the fusion protein expressed by second plasmid comprises a peptide comprising a Gal4 protein DNA binding domain, which peptide comprising a Gal4 protein DNA binding domain is bound at its C-terminal amino acid to the N-terminal amino acid of the p51 subunit polypeptide.

41. The method of any one of claims 5-8, wherein (a) the fusion protein expressed by the first plasmid comprises a peptide comprising a LexA protein DNA binding domain, wherein the p66 subunit polypeptide is bound at it's C-terminal amino acid to the N-terminal amino acid of the peptide comprising a LexA protein DNA binding domain; and (b) the fusion protein expressed by the second plasmid comprises a Gal4 peptide corresponding to amino acids 768-881 of Gal4, an influenza hemagglutinin (HA) epitope tag, and a peptide comprising six consecutive alanine residues, wherein the Gal4 peptide is bound at its C-terminal amino acid to the N-terminal amino acid of the influenza hemagglutinin (HA) epitope tag, wherein the influenza hemagglutinin (HA) epitope tag is bound at its C-terminal amino acid to the N-terminal amino acid of the peptide comprising six consecutive alanine residues, wherein the peptide comprising six consecutive alanine residues is bound at its C-terminal amino acid to the N-terminal amino acid of the p66 subunit polypeptide.

42. A method of making a pharmaceutical composition which comprises:

a) determining whether a compound inhibits HIV-1 reverse transcriptase by the method of any one of claims 1-8;

b) recovering the compound if it is determined to inhibit HIV-1 reverse transcriptase; and

c) admixing the compound with a pharmaceutically acceptable carrier.

43. A method of inhibiting formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase, which comprises contacting either (1) the p51 subunit polypeptide, (2) the p66 subunit polypeptide, or (3) both the p51 subunit polypeptide and the p66 subunit polypeptide, with an effective amount of a compound determined to do so by the method of claim 2, so to thereby inhibit formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase.

44. A method of enhancing formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase, which comprises contacting either (1) the p51 subunit polypeptide, (2) the p66 subunit polypeptide, or (3) both the p51 subunit polypeptide and the p66 subunit polypeptide, with an effective amount of a compound determined to do so by the method of claim 4, so to thereby enhance formation of a complex between the p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase.

45. A method of inhibiting formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase, which comprises contacting either (1) the first p66 subunit polypeptide, (2) the second p66 subunit polypeptide, or (3) both the first p66 subunit polypeptide and the second p66 subunit polypeptide, with an effective amount of a compound determined to do so by the method of claim 6, so to thereby inhibit formation of a complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase.

46. A method of enhancing formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase, which comprises contacting either (1) the first p66 subunit polypeptide, (2) the second p66 subunit polypeptide, or (3) both the first p66 subunit polypeptide and the second p66 subunit polypeptide, with an effective amount of a compound determined to do so by the method of claim 8, so to thereby enhance formation of a complex between the first p66 subunit polypeptide of HIV-1 reverse transcriptase and the second p66 subunit polypeptide of HIV-1 reverse transcriptase.

47. The method of any one of claims 43-46, wherein the HIV-1 reverse transcriptase is present in a subject and the contacting is effected by administering the compound to the subject.

48. The method of claim 47, wherein the compound is

administered orally, intravenously, subcutaneously, intramuscularly, topically or by liposome-mediated delivery.

49. The method of claim 47, wherein the subject is a human being, a primate, an equine, an opine, an avian, a bovine, a porcine, a canine, a feline or a mouse.

50. The method of claim 47, wherein the effective amount of the compound is between about 1mg and about 50mg per kg body weight of the subject.

51. The method of claim 50, wherein the effective amount of the compound is between about 2mg and about 40mg per kg body weight of the subject.

52. The method of claim 51, wherein the effective amount of the compound is between about 3mg and about 30mg per kg body weight of the subject.

53. The method of claim 52, wherein the effective amount of the compound is between about 4mg and about 20mg per kg body weight of the subject.

54. The method of claim 53, wherein the effective amount of the compound is between about 5mg and about 10mg per kg body weight of the subject.

55. The method of claim 54, wherein the compound is administered at least once per day.

56. The method of claim 47, wherein the compound is administered daily.

57. The method of claim 47, wherein the compound is administered every other day.

58. The method of claim 47, wherein the compound is administered every 6 to 8 days.

59. The method of claim 47, wherein the compound is administered weekly.

60. A compound determined to be capable of inhibiting formation of a complex between a p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase by the method of claim 2.

61. A compound determined to be capable of enhancing formation of a complex between a p51 subunit polypeptide of HIV-1 reverse transcriptase and a p66 subunit polypeptide of HIV-1 reverse transcriptase by the method of claim 4.

62. A compound determined to be capable of inhibiting formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase by the method of claim 6.

63. A compound determined to be capable of enhancing formation of a complex between a first p66 subunit polypeptide of HIV-1 reverse transcriptase and a second p66 subunit polypeptide of HIV-1 reverse transcriptase by the method of claim 8.

64. A composition which comprises the compound of any one

of claims 60-63 and a carrier.

65. The method of claim 4, wherein the yeast strain is CTY10-5d with the genotype MATa ade2 trp1-901 leu2-3, 112 his3-200 gal4-gal80-URA3::lexA-lacZ.

66. The compound of claim 61, wherein the compound is capable of inhibiting growth of HIV-1.

67. The compound of claim 61, wherein the compound is a nonnucleoside reverse transcriptase inhibitor.

68. The compound of claim 67, wherein the compound is not (S)6-Chloro-4-(cyclopropylethynyl)-1,4-dihydro-4-(trifluoromethyl)-2H-3,1-benzokazin-2-one, N-[4-chloro-3-(3-methyl-2-butenyloxy)phenyl]-2-methyl-3-furancarbothioamide or (S)-4-isopropoxycarbonyl-6-methoxy-3-(methylthiomethyl)-3,4-dihydroquinoxaline-2(1H)-thione.

69. The compound of claim 67, wherein the compound is a derivative of (S)6-Chloro-4-(cyclopropylethynyl)-1,4-dihydro-4-(trifluoromethyl)-2H-3,1-benzokazin-2-one (efavirenz), N-[4-chloro-3-(3-methyl-2-butenyloxy)phenyl]-2-methyl-3-furancarbothioamide or (S)-4-isopropoxycarbonyl-6-methoxy-3-(methylthiomethyl)-3,4-dihydroquinoxaline-2(1H)-thione.

70. The compound of claim 61, wherein the enhanced formation of complex is higher than an enhancement of complex formation in the presence of efavirenz at a given concentration, that concentration being in a linear range of enhancement for both compound and

efavirenz.

71. The compound of claim 70, wherein the enhanced formation of complex is at least 20% higher than the enhancement of complex formation in the presence of efavirenz at a given concentration, that concentration being in a linear range of enhancement for both compound and efavirenz.

72. The compound of claim 70, wherein the enhanced formation of complex is at least 25% higher than the enhancement of complex formation in the presence of efavirenz at a given concentration, that concentration being in a linear range of enhancement for both compound and efavirenz.

73. The compound of claim 70, wherein the enhanced formation of complex is at least 30% higher than the enhancement of complex formation in the presence of efavirenz at a given concentration, that concentration being in a linear range of enhancement for both compound and efavirenz.

74. The compound of claim 70, wherein the enhanced formation of complex is at least 1.5 times higher than the enhancement of complex formation in the presence of efavirenz at a given concentration, that concentration being in a linear range of enhancement for both compound and efavirenz.

75. The compound of claim 70, wherein the enhanced formation of complex is at least two times higher than the enhancement of complex formation in the presence of efavirenz at a given concentration, that

concentration being in a linear range of enhancement for both compound and efavirenz.

76. The compound of claim 70, wherein the enhanced formation of complex is at least three times higher than the enhancement of complex formation in the presence of efavirenz at a given concentration, that concentration being in a linear range of enhancement for both compound and efavirenz.

77. The compound of claim 70, wherein the enhanced formation of complex is at least five times higher than the enhancement of complex formation in the presence of efavirenz at a given concentration, that concentration being in a linear range of enhancement for both compound and efavirenz.

78. The compound of claim 70, wherein the enhanced formation of complex is at least ten times higher than the enhancement of complex formation in the presence of efavirenz at a given concentration, that concentration being in a linear range of enhancement for both compound and efavirenz.

79. The compound of claim 61, wherein the yeast strain is CTY10-5d with the genotype MATa ade2 trp1-901 leu2-3, 112 his3-200 gal4-gal80-URA3::lexA-lacZ.

80. The compound of claim 79, wherein the enhanced formation of the complex is at least two-fold higher than the enhancement of complex formation in the presence of efavirenz at equivalent concentration in

yeast strain CTY10-5d with the genotype MATa ade2 trp1-901 leu2-3, 112 his3-200 gal4-gal80-URA3::lexA-lacZ.

81. The compound of claim 61, wherein the compound acts at a location on HIV-1 reverse transcriptase distinct from the nonnucleoside reverse transcriptase binding pocket.

82. A composition comprising at least one compound of any one of claims 66-81 and a pharmaceutically acceptable carrier.

83. The composition of claim 82, further comprising a known NNRTI.

84. The composition of claim 83, wherein the known NNRTI is selected from the group consisting of efavirenz, UC781, HBY097, and combinations thereof.

85. The composition of claim 82, further comprising at least one nucleoside reverse transcriptase inhibitor.

86. The composition of claim 85, wherein the nucleoside reverse transcriptase inhibitor is selected from the group consisting of lamivudine, zidovudine, and combinations thereof.

87. A method of enhancing formation of a complex between a p66 subunit polypeptide of HIV-1 reverse transcriptase and a p51 subunit polypeptide of reverse transcriptase, which comprises contacting the p66 and p51 subunits with an effective amount of at least one compound of any one of claims 66-81, so as to thereby

enhance formation of a complex between the p66 subunit polypeptide and the p51 subunit polypeptide of HIV-1 reverse transcriptase.

88. The composition of claim 82, further comprising at least one HIV-1 protease inhibitor.

89. The composition of claim 88, wherein the HIV-1 protease inhibitor is selected from the group consisting of indinavir, amprenavir, ritonavir, and combinations thereof.

90. The method of enhancing formation of claim 87, wherein the HIV-1 reverse transcriptase is present in a subject and the contacting is effected by administering the compound to the subject.

91. The method of claim 90, wherein the compound is administered orally, intravenously, subcutaneously, intramuscularly, topically or by liposome-mediated delivery.

92. The method of claim 90, wherein the subject is a human being, a primate, an equine, an opine, an avian, a bovine, a porcine, a canine, a feline or a mouse.

93. The method of claim 90, wherein the effective amount of the compound is between about 1mg and about 50mg per kg body weight of the subject.

94. The method of claim 93, wherein the effective amount of the compound is between about 2mg and about 40mg per kg body weight of the subject.

95. The method of claim 94, wherein the effective amount of the compound is between about 3mg and about 30mg per kg body weight of the subject.

96. The method of claim 95, wherein the effective amount of the compound is between about 4mg and about 20mg per kg body weight of the subject.

97. The method of claim 96, wherein the effective amount of the compound is between about 5mg and about 10mg per kg body weight of the subject.

98. The method of claim 97, wherein the compound is administered at least once per day.

99. The method of claim 90, wherein the compound is administered daily.

100. The method of claim 90, wherein the compound is administered every other day.

101. The method of claim 90, wherein the compound is administered every 6 to 8 days.

102. The method of claim 90, wherein the compound is administered weekly.

103. A method of inhibiting the growth of HIV-1 comprising administering at least one compound of any one of claims 66-81, alone or in combination with a known NNRTI, in an amount effective to inhibit HIV-1.

104. The method of claim 103, wherein the conventional NNRTI is selected from the group consisting of

efavirenz, UC781, HBY097, and combinations thereof.

105. The method of claim 103, further comprising administering at least one nucleoside reverse transcriptase inhibitor.

106. The method of claim 105, wherein the nucleoside reverse transcriptase inhibitor is selected from the group consisting of lamivudine, zidovudine, and combinations thereof.

107. The method of claim 103, further comprising administering at least one HIV-1 protease inhibitor.

108. The method of claim 107, wherein the HIV-1 protease inhibitor is selected from the group consisting of indinavir, amprenavir, ritonavir, and combinations thereof.